VOLUME-9, ISSUE-6, JUNE-2020 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Orthopaedics

# ANDERSSON LESION IN ANKYLOSING SPONDYLITIS WITH PARAPARESIS: A CASE REPORT OF 2 PATIENTS

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## ABSTRACT

**INTRODUCTION:** Andersson lesions(AL) are disco-vertebral degenerative lesions in patients with Ankylosing Spondylitis(AS). Though the prevalence is low but patients with AL have high incidence of neurological deficit. Extensive lesions may present with puesodoarthosis and neuro-deficit for which often surgical interventions are necessary. This case report describes the management of 2 such patients treated with long segment posterior instrumentation and fusion with a follow up period of 5 years.

**CASE REPORT:** Both the middle aged patients had a history of long standing AS with positive HLA B27 from late adolescent age, presented with insidious onset of increased intensity of pre-existing back pain and gradual onset paraplegia. Radiographic evaluation revealed disco-vertebral degeneration with narrowing of spinal canal at D9-10 (Patient 1) and L1-2 level (patient 2). Both the patients were treated with long segment pedicular screw fixation and decompression. Posterolateral fusion in patient 1 and posterior and anterior fusion in patient 2.

**RESULT:** Complete recovery of neurology and healing was evident by 1 year. On follow up of 5 years both were clinically pain free till the last day of report. No signs of instability were seen in the adjacent segments of the fusion levels.

**CONCLUSION:** Only a few literatures are available mainly in the form of case series for AL. The reason may be due to the low prevalence or due to missed diagnosis owing to lack of clinical suspicion. Studies with increased sample size and high level of evidence such as RCT will help in the formation of proper protocol for management.

**KEYWORDS :** Andersson lesion, Ankylosing Spondylitis, Puesdoarthosis, posterior instrumentation, fusion

## INTRODUCTION:

Andersson lesion (AL) are destructive lesions of vertebral bodies and intervertebral disc in patients with Ankylosing Spondylitis (AS) as described by Andersson in 1937 [1]. The lesions are classified from A-E based on involvement of discal and vertebral surface with E being extensive lesions [2]. The prevalence rate is low from 1% to 28 % [3], [4]. Patients with AS are more susceptible to fractures secondary to osteoporosis due to decreased bone turnover in autoimmune inflammation [5]. The management of AL is difficult as these lesions can get easily missed in plain radiograph. High degree of suspicion is required for early diagnosis[5]. This case report is of 2 patients of Andersson lesion with Ankylosing spondylitis from adolescent age group with a follow up of 5 years.

Case Report 1. 60 years old male with long standing ankylosing spondylitis from adolescent age developed insidious onset of increased intensity in back pain and gradual onset paraplegia.

Normal radiograph revealed of destructive lesion at D10-11 level. MRI suggested of destruction of endplate and intervening disc at D10-11 level with narrowing of spinal canal. No history of trauma was present. Patient was started on Anti-tubercular drugs (AKT) after initial visit to general orthopedic surgeon. With progression of weakness he visited our hospital and was operated for posterolateral fusion with posterior instrumentation using pedicular screw bilaterally at D8-9 and D12 and L1 level and connected with two 5.5mm Rods and inter-transverse process bone grafting. Decompression in the form of D10-11 laminectomy was done. As patient was started preoperatively with AKT, biopsy was done which was negative for tuberculosis and metastasis. AKT was discontinued after the biopsy report.

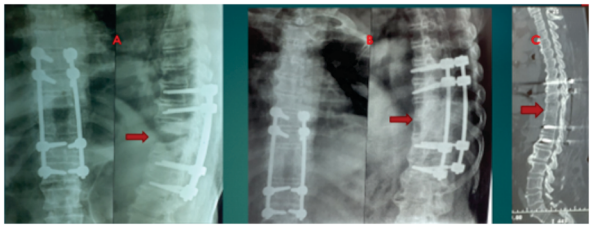

**Figure 1. Case Report 1**

A. Post-operative radiograph immediate after surgery showing long segment fixation ofAndersson lesion at D11-12 level. B. Healing with inter-body fusion seen in 5 years follow up X-Ray (AP and Lateral. C. CT scan Case Report 2. 45 years old male with long standing ankylosing spondylitis from adolescent age group developed insidious onset gradual onset paraplegia (Grade III power). The increase in back pain was not significant. Before developing AL patient was on regular physiotherapy and used to take indomethacin 75 only on acute exacerbations of joint pain. There was no history of trauma. Radiographic evaluations suggested of Andersson lesion at L1-2 level and was treated with posterolateral long segment fusion, laminectomy at L1-2 level and anterior fusion with PEEK cage and bone grafting.

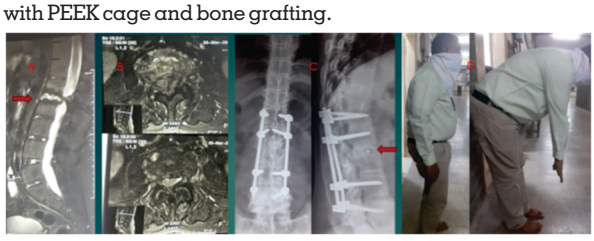

**Figure 2 Case Report 2**

A, B. Preoperative MRI (sagittal and axial view) showing

Andersson lesion with neural compression. C. Healing with inter-body fusion seen in 5 years follow up X-Ray (AP and Lateral) with long segment fixation and PEEK cage. D. Functional outcome at 5 years follow up In both the patients the Peri-operative period was uneventful without any complication. Neurology of the first patient recovered in 6 months and the second patient in 3 months with complete fusion in 1 year. On follow up of 5 years both were clinically pain free till the last day of report. No signs of instability were seen in the adjacent segments of the fusion levels.

## DISCUSSION:

Andersson lesions occur in patients with ankylosing spondylitis. Different termsfor ALhave been used in literature such as Spondylodiscitis; Destructive vertebral lesions; Spinal puesdoarthosis [6]. Etiologies are mainly divided into inflammatory and traumatic; Inflammatory being the part of natural history of AS and traumatic are puesdoarthosis following trauma [7]. Traumatic are caused due to repeated stress and biomechanical instability in fused long segments of vertebra on other ends resulting in the formation of long lever arm. This causes abnormal movements in between the two long segments leading to the formation of puesdoarthosis [8]. Bon et al described the involvement of posterior elements of the vertebral column as 'extensive lesions' which are more susceptible to puesdoarthosis [9] and thoracolumbar region(T11-L1) being the most common site [10].

Patients present with increase in the intensity and characteristics of the long existing back pain. In AS the back pain increases with rest and get relieved with activity but after development of AL the pain increases with activity [8]. There is development or increase in deformity in the back and neurological involvement is seen in 29-91% patients with AL [8-11]. As the patients have long standing back ache, the symptoms of back pain are usually ignored until the patient presents with features of neurological compressions [12]. MRI and CT scan aid in diagnosis. MRI shows increased intensity in T1 and T2 images as they are non-inflammatory lesion. T1 weighted contrast images show increased uptake in vertebral margin and in T2 imagespuesdoarthosis fluid of the same intensity of cerebrospinal fluid(CSF) is seen [13]. The MRI features often mimics tuberculosis and metastasis and due to misdiagnosis AKT are primarily started by general medical practitioners and general orthopedic surgeons [11]. Biopsy reveals features of puesdoarthosis that is fibrous tissue, small amount of callus and sclerosis of the lining vertebra [14].Surgical management is the mainstay of treatment in patients with symptomatic AL [15]. Various surgical techniques have been mentioned in the literature – anterior fusion, both anterior and posterior fusion, circumferential fusion, long and short segment posterior fusion [8], [11], [12], [15]. Study done by Ismail Shaikh et al suggest long segment fixation with posterior only fusion gives excellent results with less comorbidities as compared to anterior surgeries [8]. There is almost no concern with union as in AS due to inherent bone forming tendency [8].

## CONCLUSION:

Only a few literatures are available mainly in the form of case series for Andersson lesion. The reason may be due to the low prevalence and often due to missed diagnosis owing to lack of clinical suspicion. A more detailed study with increased sample size and studies with high level of evidence such as RCT with help in the formation of proper protocol for treatment.

## CONFLICT OF INTEREST:

None of the authors have any conflict of interest.

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